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Polio-Encephalitis Superior Acuta.

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POLIO-ENCEPHALITIS SUPERIOR ACUTA; WITH REPORT OF A CASE.¹

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B. C. D.; male; æt. 39; married; driver of milk wagon. While following his usual occupation during September, October and November, he had occasional attacks of vertigo, occurring about once a week, so severe that he was obliged to grasp a support to keep from falling. During the same period of time he also had sensations mainly in the limbs, which he describes as of an itching character, and also some feeling of numbness. In the early part of December, the attacks of vertigo became much increased in frequency, so that he had several daily, and his wife says that at this time she also noticed that he squinted, the left eye she thinks being turned inward. On December 6th, he called on his family physician, Dr. Snively, complaining some of diplopia, giddiness, frontal headache, and staggering gait. He still continued to work, and on December 13th called again on Dr. Snively, when it was noticed that the eyes were immobile and fixed in the mid position, the facial muscles had lost power and tone, and the speech was much altered. He, however, still persisted in going over his milk route daily, until, on the 15th, when in one of his vertiginous attacks, he fell out of the wagon. He then went to bed, and Dr. Snively was called next day, finding a temperature reaching 100.5; pulse increased in frequency to about 100; considerable somnolence, and a little mental disturbance, which was thought to amount to a few hallucinations. There was no activity of the bowels, and a small cathartic dose of aloin ($\frac{1}{8}$ gr.) was administered, which purged severely and continuously for an inordinate length of time. After a few days of slight fever there was free sweating, and the pulse and temperature became normal. There was about this time, some sweating of the left side of the face while the right was dry. A considerable degree of cutaneous anæsthesia of the extremities, the legs and

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forearms especially, was made out. I was called to see the patient on the 23d of December, seventeen days after he had been first seen by Dr. Snively.

I found a partial ptosis, which would probably have been complete but for the paralysis of the antagonists to the levatores, the orbicularis palpebrarum; entire immobility of the eye-ball in every direction, the eye being fixed in the mid-position, and slightly bulging; loss of power to accommodate; great dilatation of the pupil, entirely irresponsive to light (a lighted candle could be brought almost in contact with the eye, without producing the least contraction). All these symptoms were perfectly bilateral. There was also complete bilateral facial paralysis, so that he could not raise his eyebrows, close his eyes, could not blow or whistle. He found eating and drinking difficult on account of the tendency for the substances to escape through the toneless lips. The speech was nasal, muffled, and guttural, partly from paralysis of the velum palati (the uvula deviating very slightly to the left) and largely from paralysis of the levatores alæ nasi, which was shown when he pushed up the nasal cartilages during phonation, his speech then being markedly more distinct.

Smell had been lost according to his statement, during the first few days he was in bed, but had already been recovered. Taste also had been completely lost, but now also had partially recovered. I found on testing that on the left side of the tongue, taste was normal on the anterior part, and on the posterior part, paræsthetic. He would mistake salt for sugar, or vinegar for salt, and so on. On the right half of the tongue the sense of taste was wholly absent.

He had a feeling of numbness in both arms and legs, combined with a "pins and needles" sensation. I found decided but not complete anæsthesia and analgesia on the outside and back of right leg, and on the inside of left, as well as on the ulnar and posterior surfaces of both forearms. There was great pain on pressure over the fourth lumbar spine, and also some spontaneous pain in this region.

The plantar, the cremasteric and the abdominal reflexes were all diminished, especially the plantar on the left side. The knee jerks were absent, even on reinforcement. There were spontaneous movements on holding out the limbs and a coarse tremor of all four extremities on voluntary motion. He sways on standing

with eyes open, very much more with eyes closed. His gait is very irregular, and ataxic, especially marked on turning in his course. No marked loss of power could be determined in any muscle, or set of muscles, in the extremities.

He has never had syphilis, and never used alcohol; he chews tobacco excessively and smokes some. He has an aunt living who is paralytic, and another aunt and an uncle have died of paralytic affections.

My friend, Dr. J. A. Cramp, was asked to examine the eye grounds, and to test the visual fields. He reports as follows:

My dear Dr. Wolfe:

I examined Mr. Daniels as you requested. External examination showed complete paralysis of all the ocular muscles (bilateral), the pupil was dilated and would not respond to light. Accommodation was paralyzed. There was ptosis principally on left side; the patient's distance vision was fair, being $\frac{5}{6}$; no contraction of the field of vision. The ophthalmoscope showed no fresh zones of inflammation in neither the retina, choroid, nor nerve; although the nerve borders above and below were slightly obscured by connective tissue; this condition I would state, we often find in hypermetropia, which defect the patient had to the extent of about two dioptries.

On the 3d of January, eleven days after the above condition was found, I found that the pupil responded readily to light impressions and was not greatly dilated. Power of accommodation had returned so that he could read. The ptosis had largely disappeared. Taste had fully returned, and the anæsthesia of the legs had disappeared, but persisted in the arms. His gait was markedly less ataxic. The other symptoms were unchanged, except that a very slight improvement in his speech may be noted.

On the 19th of January, twenty-seven days after my first examination, I found the ptosis entirely gone, the pupils normal in size and reaction, a little spasmodic lateral movement of the eyes, usually of one alone, on attempting to follow an object. He could now raise his eyebrows, shut his eyes, blow, whistle, retain food within his lips, and could give some facial expression to his emotions, although in this respect, power was not proportionate to that of voluntary motion of the facial

muscles. The anæsthesia has disappeared, but there is an irregular paræsthesia of trunk, which he defines as a feeling of a hot cloth, lying on a patch of skin, now on the abdomen, now on the chest, and again on the back, but not constantly present in any situation.

He now stands well with eyes open or closed, but still is slightly ataxic in gait. There is slight inco-ordination of left arm, shown on touching his nose, and a coarse tremor of all four extremities. The left knee jerk is very slightly present, and develops into an almost normal one, on reinforcement. The right is still altogether absent.

On the 22d of January, when seen for the last time before the conclusion of this paper, he could move his eyes inward, upward, and downward to a considerable extent, but outward movement was still entirely absent. He could now smile broadly, and walk almost without any noticeable inco-ordination, with the exception of a halt before starting, in turning half way round. The sixth nerve seems the last to recover, and here there will probably remain some residual paralysis.

The treatment pursued in the case from the time I saw him, was potass iodid gr. $\times$ t.d., until about the third week, when $\frac{1}{50}$ gr. doses of strychnia sulph. were given in addition, three times a day. He was kept in bed, and all mental excitement and exercise were prohibited.

The chief, if not the exclusive lesion in this case, I believe was in the nuclei of the 3d, 4th, 6th, and 7th cranial nerves. The paræsthesia of the extremities, which existed for three months before the cranial nerve symptoms appeared, and the occasional attacks of vertigo that appeared during the same time, point to the possibility of some vague degenerative disease in its earlier stages, but the anæsthesia and analgesia with the ataxia, which appeared in conjunction with the major symptoms of the main attack seem to me to point to involvement of the tegmentum, or probably the thalamus. This involvement seems to me to be indirect, most likely due to circulatory or nutritional disturbance of some kind, while the direct lesion was inflammatory in nature, and in-

volved the motor cells of the nuclei of the nerves already mentioned, just as an anterior polio-myelitis has the inflammatory action limited to the motor cells of the anterior cornua. Spasm and ataxia manifested in the gait, it should be remembered, were symptoms which entered constantly into the clinical picture of polio-encephalitis superior acuta, as observed by Wernicke, and their association in the present case with a history of paræsthesia, affecting the same parts for some time previous to their development, might be looked on as purely incidental. The topographical relations of the nuclei of the 3d, 4th, 6th, and 7th nerves to the tegmentum, are such that a focal lesion of the former, might be expected to affect the latter in such a way as to produce indirect symptoms pertaining to sensory conduction, while the crusta and consequently the pyramidal tract might easily escape. Again, a focus of inflammation involving the nuclei, would most naturally involve also the contiguous parts, and thus the symptoms might be direct. The order of recovery, however, the sensory phenomena being the first to disappear, argues in favor of the former view.

As to the nature of the lesion, the view that it was inflammatory is tenable on the ground of gradual onset, slight rise in temperature and acceleration of pulse, and the occurrence during the prevalence of an epidemic of influenza. I need not dwell upon what has been a matter of such universal observation, the tendency of the toxine of *la grippe* to produce lesions of both the peripheral and central parts of the nervous system. That some such agency is active in the production of acute anterior polio-myelitis, and probably of some other acute system diseases of the nervous tissues is a theory which is undoubtedly gaining in favor from year to year.

The transient loss of smell, and the somewhat more persistent loss of taste, are not very easily explained. It may be worth while to consider that the uncinate gyrus, the hippocampal gyrus and the anterior commissure and cornu ammonis are all parts whose topo-

graphical relations to the nuclei certainly affected, are such that they sustained more or less liability of being indirectly, if not directly, affected. The transient nature of these symptoms, too, was such that, it may be assumed that structures of a different nature, as the cortical cells, were affected, or that the symptoms were of an indirect nature. In regard to the affection of taste, the view of Gowers, that all the taste fibres arise from the nucleus of the fifth, must be borne in mind. The absence of all other sensory and motor symptoms connected with this nerve, however, militates strongly against this view of the nature of the affection. Somewhat more plausible argument may be made in favor of an implication of the fibres of the chorda tympani, through nutritive changes, brought about by their close relation to the facial fibres, proper. That these fibres (the facial) underwent trophic changes of a decided character is, of course, simply in accordance with one of the facts most thoroughly known, in the pathology of nerve degeneration. That such intense changes might exercise a deleterious effect upon other very closely associated structures is by no means an altogether unwarrantable assumption. I, however, incline to believe that in the affections of both taste and smell the origin was cortical rather than nuclear or peripheral.

The diplegia facialis is a symptom, which, heretofore, has not been observed in this disease. When we consider the close relations of the nucleus of the 6th and 7th nerves, there being most probably communicating fibres between the two, and the still closer relation of the root fibres of the facial to the nucleus of the 6th, it seems rather remarkable that it should be the rule that the facial escapes. The probable origin of the fibres which supply the levator palpebræ, from the nucleus of the 7th establishes a still further close relation between the oculo-motor nerves and the facial. It will be seen, then, that the present case is, in all respects, one of the most completely typical yet observed. The involvement of the three branches of the facial, since we are able posi-

tively to exclude a peripheral lesion, at once places this focus in the nucleus. The complete and persistent loss of power of expression, also, fixes the lesion below the optic thalamus, unless, indeed, we may consider that organ implicated. At all events, the cortex and the fibres of the corona radiata, as well as the intra-capsular course of the tract can positively be excluded.

The completeness of the ophthalmoplegia at once removes the disease from the chronic type which has been so well studied by Von Graefe. In this the pupil constrictor and the ciliary muscle are not involved. Besides this we have a series of concomitant phenomena, including a specific poison, acting as a cause which place the disease in the acute class. The order of recovery is interesting, since those symptoms disappear first, that never appear at all in the chronic variety, indicating that a less intense cause is sufficient to affect the extrinsic muscular apparatus, than would suffice for the intrinsic.

The tendency to recovery in this case, is also peculiar, since the type described by Wernicke is almost invariably fatal. There is, however, in this, a still further analogy to the prototype, acute anterior poliomyelitis, which again counts in establishing this case as particularly typical. Should there remain, as I think there will, some residual paralysis, this observation will be still more strongly confirmed.

The study of this disease has been so much limited, that its existence as a pathological entity is doubted by many neurologists. What observation there has been is probably confined entirely to Germany. Instances, therefore, are rare. The present case, it would seem, should go far in establishing its rank as an independent disease. The chronic progressive variety occurs more often, and its existence and identity are well established.

To summarize and conclude: I have in this paper reported a case of inflammation of the motor cells of the nuclei of the 3d, 4th, 6th and 7th cranial nerves, of infectious origin, in which the initial symptoms were diplo-

pia, strabismus and vertigo ; the established symptoms directly due to the lesion, external ophthalmoplegia, cycloplegia, iridoplegia (mydriasis) and ptosis with diplegia facialis giving rise to great alterations in speech ; and with loss of smell and taste, inco-ordination, and sensory disturbances as indirect symptoms ; the entire complement of symptoms being established in the course of less than three weeks, after a duration of about ten days progressive recovery ; classified as polio-encephalitis superior acuta, presenting probably the most completely typical case yet recorded.

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